electrophysiologic studies. This noninvasive technique detects low-amplitude, high-frequency signals at the terminal portion of the QRS complex. In patients with syncope, the presence of these signals appears to have a sensitivity and specificity of 80% to 90% for eliciting serious ventricular arrhythmias by electrophysiologic studies.

Although frequently ordered, ambulatory Holter monitoring may not define an exact cause of syncope because of the lack of well-established criteria for interpreting abnormal results. To determine a specific cause for syncope, the recorded arrhythmia must coincide with the patient's symptoms, but this occurs in only 3% to 5% of cases. Some arrhythmias are common among the general population. For example, bradycardia (less than 40 beats per minute), brief runs of supraventricular tachycardia, premature ventricular contractions, and even multiform or paired premature ventricular contractions have been recorded in substantial percentages of asymptomatic ambulatory patients. The optimal duration of monitoring has also not been determined. Patient-activated intermittent recorders, loop recorders, and transtelephonic electrocardiographic recordings are useful alternatives in patients in whom an arrhythmic cause is strongly suspected.

In patients without known heart disease, the head-up tilt test can be useful in diagnosing vasovagal syncope. This noninvasive procedure involves tilting a patient at 60 to 80 degrees for 10 to 60 minutes with or without an infusion of isoproterenol hydrochloride. A positive response reproduces the patient's symptoms in association with marked bradycardia, hypotension, or both. The mechanism for this faint involves the Bezold-Jarisch reflex. Some patients may respond to treatment directed at blocking certain aspects of this reflex arc (for example, β-blockers to blunt the endogenous catecholamine release).

The prevalence of psychiatric disorders in patients with syncope may be much higher (as high as 24%) than previously thought. Psychiatric syncope appears to consist predominantly of panic disorders and major depression. These patients tend to be younger; report more prodromal symptoms such as lightheadedness, shortness of breath, dizziness, and palpitations; and experience syncopal symptoms almost weekly. The mechanism of psychiatric syncope is probably multifactorial and ranges from hyperventilation to vasovagal syncope. Diagnosing and treating the psychiatric disorder may alleviate syncopal symptoms in some patients.

In the 1980s, a considerable percentage of patients were left with a diagnosis of "syncope of unknown origin." In the 1990s, physicians have an array of new diagnostic tests to select from and use to decipher the exact cause and treat patients who present with syncope.

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Primary Human Immunodeficiency Virus Infection—Will You Miss the Diagnosis?

THE PREVALENCE of human immunodeficiency virus (HIV) infection continues to increase. Worldwide, 9 to 11 million people are infected with HIV; by the year 2000, 40 million people may be infected. A new, unpublished study by the San Francisco Department of Health found a higher prevalence of HIV antibodies among gay men in the 17- to 25-year-old group compared with older gay men. According to the Centers for Disease Control, the number of cases of the acquired immunodeficiency syndrome (AIDS) associated with heterosexual transmission of HIV has been increasing steadily. The number of AIDS cases in drug injectors, women, and children is also on the rise. Most seroconverters—53% to 93% of homosexual men with new HIV infection—will have symptoms of primary HIV infection, and most (87%) will seek medical attention. These facts indicate that physicians will continue to see patients who have symptoms of acute HIV infection.

Recent studies have shown that primary infection with HIV is associated with high plasma levels of virus, enhancing potential infectivity. In 1992, people at risk for infection will come from two categories of transmission: sexually transmitted and blood-borne. New cases of acute HIV infection will occur in heterosexuals and homosexuals with multiple sexual contacts and in sexual partners of persons at risk, especially intravenous drug users. Blood-borne infection remains a risk for fetuses, neonates, drug injectors, and, less so, for transfusion recipients and health care workers with needle-stick injuries. The early recognition of primary HIV infection is important to allow counseling to prevent transmission and to begin early treatment and monitoring that can increase the length and quality of life.

Primary HIV infection causes a mononucleosis-like syndrome, with or without aseptic meningitis, associated with seroconversion for the HIV antibody. The incubation period before clinical illness has ranged from five days to three months. Clinical manifestations are of sudden onset, may last 3 to 14 days, and include fever, pharyngitis, lymphadenopathy, arthralgia, myalgia, headache, retro-orbital pain, nausea, vomiting, diarrhea, anorexia, oral thrush or ulcerations, and an erythematous maculopapular rash. Neurologic manifestations include meningoencephalitis, myelopathy, peripheral neuropathy, and the Guillain-Barré syndrome. The syndrome can mimic influenza, rubella, infectious mononucleosis, toxoplasmosis, viral hepatitis, syphilis, herpes simplex infection, or aseptic meningoencephalitis. Laboratory evaluation at this stage is frequently not diagnostic. Abnormal liver function test results, especially elevated alkaline phosphatase and aspartate aminotransferase levels, have frequently been reported. The lymphocyte count declines initially, followed by an inversion of the CD4/CD8 ratio and a progressive decline of CD4 cells.

The antibody test for HIV using enzyme immunoassay can be negative, making early diagnosis difficult. One recent example is a 39-year-old heterosexual man who had been attending "sex orgies" with multiple partners, and six weeks later severe headaches, neck stiffness, hypersomnolence, myalgias, generalized weakness, malaise, anorexia, and weight loss developed. After a two-week illness, he sought medical attention. Examination revealed diffuse adenopathy and nuchal rigidity; the results of routine laboratory tests

were unrevealing. His initial HIV antibody test by enzyme-linked immunosorbent assay (ELISA) was negative.

A diagnosis of primary HIV infection depends on the detection of specific viral products or seroconversion. One method is an assay for the p24 antigen, a core protein of HIV-1. In the first few weeks after infection, there is an initial burst of virus replication with high serum levels of p24 antigen and plasma viremia. An HIV antibody response, however, may occur as early as 3 weeks or as late as 42 months after exposure to the virus. Screening for the HIV antibody is usually done using the ELISA test. In most laboratories, the more specific Western blot test will be done only to confirm a positive ELISA test but, in fact, the Western blot test may be positive days to weeks earlier. In patients with symptoms suggestive of acute HIV infection, it is important not to rely solely on the ELISA but to also request the Western blot and p24 antigen assay. In the case noted, when the patient was first seen, the initial Western blot result was indeterminate and the p24 antigen test was negative. The p24 antigen and Western blot tests were positive nine days later, but the ELISA test for HIV-1 antibody continued to be negative for four more days. Viral culture and the use of the polymerase chain reaction to detect HIV DNA sequences can also be diagnostic, but these tests are neither cost effective nor readily available. If an acute illness consistent with primary HIV infection remains undiagnosed, it is important to repeat the serologic tests to detect late seroconversion. Depending on the individual case, it would be reasonable to repeat the Western blot and p24 antigen assays weekly during the acute illness and monthly thereafter if the illness remains undiagnosed.

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Pressure Support Mechanical Ventilation

PRESSURE SUPPORT mechanical ventilation, first introduced ten years ago, is widely used and is now incorporated into most late-model ventilators, such as the Puritan-Bennett 7200 and the Siemens Servo 900C.

Pressure support assists spontaneous breaths initiated by patients on mechanical ventilators by maintaining a constant preset airway pressure throughout the breaths. Because the constant pressure is maintained only until inspiration stops, the patient determines both tidal volume and respiratory rate. Flow is initially high to reach the preset pressure rapidly, but it then slows continuously. In contrast, conventional assisted breathing maintains a constant inspiratory flow until a preset volume is reached; pressure increases continuously throughout the breath.

"Low-level" pressure support, with preset pressures of 4 to 8 cm of water, does the work necessary to overcome resistances of the endotracheal tube and ventilator circuit, decreasing the patient's work to approximate breathing without them. "High-level" pressure support, using preset pressures of more than 10 to 15 cm of water, decreases a patient's work further; pressures as high as 20 cm of water may be necessary to prevent fatigue.

Pressure support has many advantages over conventional assisted ventilation. It automatically limits peak airway pressures to a safe level. At any assist mode, pressure support decreases inspiratory muscle work and oxygen consumption by "pushing" gas into the lungs, decreasing a patient's need to "pull" gas in. The magnitude of this effect on work is dependent on the preset pressure level, which determines the ventilator flow rate and the ventilator's "push." Because a built-in microprocessor slows the flow rate progressively as alveolar pressure rises, there is better filling of poorly communicating portions of the lung. This improves ventilation-perfusion mismatch and oxygenation and increases lung distensibility and tidal volume. Because peak airway pressures are lower, cardiac output decreases less than with conventional assisted ventilation. For these reasons, and possibly because patients set their own breathing pattern, pressure support often provides greater patient comfort and thereby requires less sedation than either unassisted breaths or conventional assisted breaths. Finally, because the inspiratory muscles continue to do some work throughout inspiration, pressure support can provide graded muscle training during weaning trials.

Disadvantages include a need for more careful monitoring because minute ventilation may change if either lung resistance or compliance changes spontaneously; a lack of backup, unless used with intermittent mandatory ventilation; the failure of a breath to turn off if there is a leak in the system; and the need to provide additional pressure equal to the patient's positive end-expiratory pressure. Because the patient must trigger the ventilator, pressure support cannot be used to correct respiratory depression from drugs or to manage neuromuscular respiratory failure caused by muscle disease or disuse atrophy or fatigue.

It is desirable to assist spontaneous breathing with low-level pressure support when using intermittent mandatory ventilation or synchronized intermittent mandatory ventilation, because high inspiratory muscle work, oxygen consumption, and fatigue during spontaneous breaths may disastrously exceed even those during spontaneous breathing without a ventilator. Pressure support may also substitute for T-tube trials during weaning, possibly better simulating conditions following extubation.

Pressure support pressures not high enough to prevent fatigue are associated with palpable contraction of the sternocleidomastoid muscles; the pressure support should be increased until they are no longer palpable. Definitive information on whether pressure support is preferable to other methods for weaning is not yet available; anecdotal and physiologic evidence suggests that it is. Extubation is usually possible when a patient tolerates support of 4 to 8 cm of water, the pressure required to overcome equipment resistance.

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